

Fluorodeoxyglucose positron emission tomography-computed tomography findings in a case of xanthogranulomatous pyelonephritis

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ABSTRACT

Xanthogranulomatous pyelonephritis (XGNP) is an uncommon condition characterized by chronic suppurative renal inflammation that leads to progressive parenchymal destruction. This condition can clinically present as recurrent urinary tract infections, flank pain, hematuria, and occasionally sepsis, and weight loss. This condition is usually associated with obstructing renal calculus. We present 18-fluorodeoxyglucose positron emission tomography-computed tomography (18-FDG PET/CT) findings in an elderly male suffering from pyrexia and weight loss and suspected urinary tract infection. PET/CT findings in this case lead to diagnosis of XGNP. This diagnosis should be kept in mind while evaluating similar symptoms and PET/CT scan findings.

Keywords: 18-Fluorodeoxyglucose positron emission tomography-computed tomography, urinary tract infection, xanthogranulomatous pyelonephritis

INTRODUCTION

The use of 18-fluorodeoxyglucose positron emission tomography-computed tomography (18-FDG PET/CT) in the field of oncology is well-established; however, 18F-FDG is not tumor specific. Aside from physiological uptake FDG also may accumulate in benign processes. Knowledge of these FDG-avid non-malignant lesions is essential for accurate PET interpretation. [1] Also this non-specificity of FDG uptake is being successfully used in evaluating infective, inflammatory diseases and pyrexia of unknown origin. [2]

We describe use of FDG PET/CT for evaluation xanthogranulomatous pyelonephritis (XGNP), a benign, chronic suppurative renal inflammation.

CASE REPORT

A 60-year-old Indian man underwent 18-FDG PET/CT for



evaluation of pyrexia and weight loss of 9 kg in 2 months. His blood examination revealed anemia, leukocytosis, and high erythrocyte sedimentation rate. Patient's serum creatinine was found to be raised (1.9 mg%, normal up to 1.4 mg%). His chest X-ray was normal. He was found to have multiple calculi in left kidney on ultrasonography (USG). USG also demonstrated multiple hypo echoic masses; thinned out parenchyma; and a dilated collecting system suggesting a chronically obstructed kidney. Suspicion of pyelonephritis was raised in the USG report and rest of the abdominal organs were unremarkable. Urinalysis showed pyuria and Escherichia coli was detected in urine culture. Antibiotic therapy was instituted for treatment of the urinary tract infection. Even after extensive antibiotic therapy, the fever remained unresolved and weight loss continued. Before invasive diagnosis, patient was referred for a whole body 18-FDG PET/ CT to confirm kidney as the sole site of active disease and rule out any extra-renal pathology as the cause of symptoms.

For PET/CT, 10 mCi of FDG was injected intravenously to patient after 6 h of fasting. After 1 h of injection, patient was scanned on dedicated 16 slice PET – CT (GE – STE 16). Whole body CT scan was obtained as part of PET/CT protocol on a multi-slice CT with 3.5 mm slice thickness without contrast injection. Contrast administration was withheld considering the increased serum creatinine level. Standardized uptake value (SUV) of FDG was normalized to body weight obtained over lesions.

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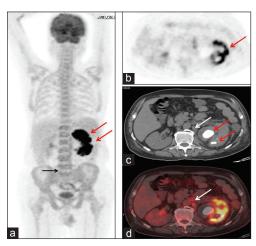


Figure 1: The Maximum intensity projection image [Figure 1a and b, arrows] revealed intense fluorodeoxyglucose (FDG) uptake in cortex of grossly enlarged, hydronephrotic left kidney. Standardized uptake value (SUV) max of uptake was 14.1. Multiple calculi were seen in pelviureteric junction and in pelvi-calyceal system [Figure 1c red arrows]. Extensive perinephric fat stranding and mild FDG uptake in perinephric, retroperitoneal lymph nodes was seen with SUV max of 4.1 [Figure 1c and d white arrows]. The findings were reported as possible xanthogranulomatous pyelonephritis. This diagnosis was confirmed post-nephrectomy

The maximum intensity projection (MIP) image [Figures 1a and b, arrows] revealed intense FDG uptake in cortex of grossly enlarged hydronephrotic left kidney. SUV max of uptake was 14.1. Multiple calculi were seen in pelviureteric junction and in pelvi-calyceal system [Figure 1c red arrows]. Extensive perinephric fat stranding and mild FDG uptake in perinephric, retroperitoneal lymph nodes was seen with SUV max of 4.1 [Figure 1c and d white arrows]. The findings were reported as possible XGNP. In addition to these findings diffuse FDG uptake was seen in bone marrow [black arrow in Figure 1a]. This could be due to anemia or the pyrexic state. Left nephrectomy was performed and histopathology confirmed XGNP. Post-nephrectomy, patient's fever resolved and he reported gradual weight gain over last 4 months.

DISCUSSION

XGNP is an uncommon condition characterized by chronic suppurative renal inflammation that leads to progressive parenchymal destruction. The disease process is characterized by the destruction and replacement of renal parenchyma by lipid-laden macrophages (xanthoma cells).[3] This process is usually diffuse but rarely may be focal. The patients present with recurrent urinary tract infections, flank pain, hematuria, and occasionally sepsis and weight loss. [4] The main predisposing factor appears to be obstruction, and an obstructing renal calculus is present in up to 75% of cases. It is postulated that renal obstruction promotes recurrent renal tract infections that lead to an abnormal immune response causing parenchymal destruction. [5] Whereas the first-line treatment of choice for both, localized and diffuse XGNPs, is conservative (appropriate antibiotic treatment), the approach to surgical treatment is different: Nephron-sparing surgery is warranted in focal XGNP, but diffuse XGNP will require nephrectomy with resection of all other involved tissue in most cases. Early diagnosis and prompt treatment play a crucial role in minimizing the morbidity and mortality rates of XGNP.[6]

The typical CT findings are unilateral renal enlargement and parenchymal inflammation. Multiple areas of low attenuation are visualized within the kidney, representing dilated renal calyces and pus-filled cavities replacing destroyed renal parenchyma. CT of our case scan had similar findings. CT is also very useful in determining the extent of the inflammatory process in perirenal tissues including the iliopsoas muscle, abdominal wall, diaphragm, skin, and bowel.^[7]

Though XGNP can be suspected on CT findings alone, with rising use of PET/CT in evaluation of pyrexic states, added knowledge of PET findings can be advantageous to add certainty to the diagnosis. Scarce data is available on FDG PET/CT findings in XGNP, in form of case reports. [8,9] CT findings characteristic of XGNP along with diffuse FDG uptake in kidney and mild FDG uptake in retroperitoneal lymph nodes is seen in the diffuse XGNP. [8] Mild FDG uptake in retroperitoneal lymph nodes and extensive perinephric fat stranding are suggestive of benign inflammatory pathology in such cases. Our case has similar PET/CT findings. However it should be kept in mind that focal XGNP can mimic pathologies such as pyelonephritis, tuberculosis, renal abscess, renal cell carcinoma, and renal metastasis on PET/CT. [9] Thorough clinical history, and correlation with anatomical imaging are essential components to arrive at diagnosis of XGNP.

To conclude, we present FDG PET/CT findings in XGNP. Because, use of FDG PET/CT for evaluation of infective and inflammatory conditions is on the rise and such condition can coexist in patients with known malignancy, this possible diagnosis should be kept in mind, when similar uptake pattern and CT findings are encountered in practice.

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